pulmonary embolism and died three hours later. An embolus occupied the pulmonary artery, resembling a blood clot found in the left common and internal iliac veins. Hastings also states that in a patient who died from pulmonary embolism after an operation a thrombus occupied the right cardiac ventricle, and he thought it possible that this intraventricular clot furnished the embolus.

We must bear in mind that individuals apparently in good health die suddenly in the street, in the arm-chair, in a bath, or even during sleep; it is a fair assumption that some of the instances of sudden death occurring during convalescence from surgical operations may be due to failure of the heart absolutely unconnected with the operation. It is, however, undeniable that thrombosis of the pelvic veins after ovariotomy or hysterectomy is a source of fatal emboli.

At present there is very little evidence available as to the cause of the thrombosis, but it can scarcely be doubted that sepsis—it may be only of a mild type—is responsible for some of the cases.

A careful consideration of the matter reveals beyond any doubt that pulmonary embolism occurs much more frequently after hysterectomy for fibroids than after any other operation, and it is especially liable to happen in women who are profoundly anaemic from profuse and prolonged menorrhagia. This indicates that long-continued and irregular losses of blood induce some change in the composition of this important fluid which favours its coagulation.

It is probable that the thrombosis is due to a combination of causes, such, for example as sepsis, cardiac disease, chill and exposure, especially in prolonged and difficult operations, damage to the walls of blood vessels, and circulatory disturbances due to anaesthetics.

It has been suggested that the practice of keeping patients strictly confined to bed for two or three weeks after hysterectomy, and allied operations, is responsible for the thrombosis which is the source of these fatal emboli. Some American surgeons act on this suggestion, and insist on their patients getting out of bed a few days after such operations. This method does not commend itself to British surgeons. In my own practice I make it a rule, even in the most favourable conditions, to keep the patients confined to bed for two weeks. No patient is allowed up until her temperature has been normal for at least three days.

In cases of embolism of the pulmonary artery death does not always occur immediately, but may be postponed for an hour or more after the lodgement of the embolus. On one occasion I saw a patient within ten minutes of the lodgement of a pulmonary embolus. The patient, a woman 35 years of age, was livid but did not breathe, yet her heart beat regularly and forcibly for five minutes after my arrival, then stopped suddenly.

Trendelenburg is of opinion that in some of these cases it might be possible to remove this clot by surgical intervention. After careful consideration of the matter he carried out this operation on a woman aged 63 years. He raised an osteoplastic flap on the left side of the thorax, exposed the conus arteriosus, and intended to withdraw the clot through a slit in its walls by means of a specially-constructed pump. The patient died from excessive bleeding before the clot could be extracted. The operation was hindered by an adherent pericardium.

When patients who are profoundly anaemic from menorrhagia due to fibroids undergo hysterectomy, it is a useful measure to give them 20 grains of sodium citrate twice daily in order to diminish the abnormal tendency of the blood to coagulate in the vessels. Certainly this drug should be administered if there is the least evidence of thrombosis. I have seen good consequences follow its use.

In conclusion let me reiterate that the history of hysterectomy, as a radical method of treating uterine fibroids, during the last ten years has been a record of continuous improvement in the hospitals of London. Like all valuable operations, especially those connected with the reproductive organs, hysterectomy for fibroids has had to pass through a probational period of prejudice. To-day no major operation can show so small a mortality, and what is more to the point, no major operation produces such permanent good effects.

Surgery can now enable a wife suffering from "an issue of blood," and spending half her life as a chronic invalid, to become a companion to her husband; or, a spinster afflicted with a bleeding fibroid, and living in humble circumstances, to earn her own living, and cease to be a domestic incubus.

It is a great gratification to realize that hysterectomy for fibroids occupies in London to-day an established position in surgery from which no misrepresentation can raze it.

REFERENCES.

[1] *Journal of Obstetrics and Gynaecology of the British Empire*, May, 1908. [2] Ibid., 1906, x, 1. [3] *Lancet*, 1901, i, 452. [4] *Archives of the Middlesex Hospital*, 1907, xi, 78.

THE ETIOLOGY OF RICKETS: A CLINICAL AND EXPERIMENTAL STUDY.*

By LEONARD FINDLAY, M.D.,

ASSISTANT TO PROFESSOR OF PATHOLOGY, AND CLINICAL TUTOR WITH PROFESSOR OF CLINICAL MEDICINE, GLASGOW UNIVERSITY; EXTRA DISPENSARY PHYSICIAN, WESTERN INFIRMARY, AND EXTRA HONORARY PHYSICIAN TO OUTDOOR DEPARTMENT OF ROYAL HOSPITAL FOR SICK CHILDREN, GLASGOW.

[WITH SPECIAL PLATE.]

MANY and varied have been the causes assigned for rickets. Almost every prejudicial circumstance in the life of the infant—and in that of the parent, too, for that matter—has been cited as the etiological factor.

ETIOLOGICAL THEORIES.

According to Kassowitz[1] and some of the German school, it is of congenital origin, a theory, however, mainly founded, at least by its earlier adherents, on macroscopic appearances alone, and not on the finding of any characteristic histological changes in the bones. In every infant the bones are soft, the abdomen may be protuberant, the fontanelles will be open, and there will be some swelling of the costo-chondral junctions, and yet it was on the ground of these facts alone that such a theory was based. The later advocates[2] of this theory, however, did examine the minute histological changes in the bones, but as much of the material studied was obtained from stillborn children, and as the pathological anatomy of osteochondritis syphilitica and rachitis was at that time only imperfectly understood, these two conditions were much confused, and it is not improbable, therefore, that many of the supposed rachitic lesions were in reality of a syphilitic nature. As these two processes came to be differentiated from one another, each succeeding published record of work done on this subject showed a marked diminution in the proportion of congenital rickets. Kassowitz in 1882 considered that 80 per cent. of all children born were rachitic; in 1897 Tschistowitsch[2] put the proportion at 12 per cent., while more recently (1902) Escher,[3] after investigating a large series of cases both clinically and histologically, was only able in one single instance to make a diagnosis of congenital rickets.

There is of course such a condition known and described as "fetal rickets," but it is considered by the majority of observers an entirely different disease to what ensues after birth, and is, moreover, as rare as ordinary rickets is common. It will probably be better then, at least for the present, to classify it apart, and designate it by one of its pseudonyms—for example, "osteogenesis imperfecta."

Closely allied to the above theory (congenital origin of rickets) is that mainly advocated by Siegert,[5] who holds that the condition is hereditary, and dependent on some inherited weakness or predisposition. This after all is merely a case of begging the question and failing to discuss the main points at issue. Hausen,[4] however, reports the case of a stallion who begot seven rachitic foals. Later two of the mares, one of which had given birth to three and the other to two of these rachitic foals, became pregnant to another stallion, and bore healthy non-rachitic offspring.

It is during extrauterine life, however, that most present-day authorities believe the etiological factors play their part, and the general consensus of opinion would ascribe the disease to incorrect feeding, bad hygiene, and want of sunlight. Of all these possible malign influences it is some error in feeding which receives most credence and support.

* The work was carried out in the Pathological and Physiological Laboratories, University of Glasgow.

By some it is said to be due to an entire want of suckling, to the mother not nursing the child long enough, to suckling at too frequent intervals, and by others, again, to prolonged lactation. Siegert[6] found that out of 845 artificially fed infants 81 per cent. developed rickets, while in 923 infants who had been nourished at the breast for at least 4½ months, only 31½ per cent. became rachitic. On the other hand, Holt[7] mentions that although negro and Italian children in New York are entirely breast-fed, they are exceedingly prone to develop the disease.

In artificially fed infants, and in the feeding of children, almost every possible quality and combination of the different constituents have been accredited with the power of inducing the disease. Some say it is consequent on a deficiency of proteid, others on an excess of carbohydrate, but perhaps the majority consider that it is due to a deficiency of fat and proteid with an excess of carbohydrate. This is a combination of factors, which, it must be remembered, may be present in the food of both naturally and artificially fed infants. According to Holt and Cheadle,[8] it is in the presence of a low percentage of both fat and proteid that rickets is liable to result, while Bland-Sutton[9] teaches that it is entirely due to a deficiency of fat. The latter's well-known experiments in the London Zoological Gardens with the lion cubs and young monkeys lend great support to his view. By only one change in the general régime of these animals, namely, the addition of fat and cod-liver oil to their diet, lion cubs were reared free of rickets for the first time in ten years. Ashby,[10] while lending his support to the idea of a low percentage of fat and proteid, considers that it is not improbable that fermentation of the carbohydrates may produce toxins, which are responsible for at least some of the phenomena of the disease. Heitzman[11] considers that "lactic acid" is the baneful product, and claims to have induced the condition experimentally by its administration. He believes that the lactic acid results from the fermentation of the carbohydrate and irritates the ossifying tissue, but other observers have not been able to confirm this formation of lactic acid, and have failed to bring about rachitic changes by its administration.

Esser,[12] who has lately written on the subject, believes that the disease is due to over-feeding, and bases his theory mainly on the similarity of the blood pictures in rickets and chronic over-feeding. He contends that over-feeding produces a chronic gastro-enteritis, and results in a deficient absorption of the food constituents. Cheadle,[13] too, in one place speaks of rickets ensuing on starvation, which is, however, contrary to clinical experience.

Considering the fact that the rachitic changes in the bones are in great part characterized by deficient calcification, it is not surprising that some observers have looked for the etiological factor in a defect of lime metabolism. It has been attributed to an absence or deficiency of lime in the food, to a defective anabolism of the lime salts, and, lastly, to an increased katabolism of these same salts—in short, to all the possible variations in the process of calcification. Chossat[9] produced curvature of the bones experimentally by depriving animals of earthy salts, but Friedleben doubted the condition being true rickets. Voit[14] also believes that he was successful in causing rickets by depriving animals of calcium. Katz[15] saw typical rickets in a fowl which had been confined in a narrow cage for some months, and which he considered due to the fact that the bird could not get a sufficient amount of lime. Reimers and Boye[16] also noted some changes in the bones of dogs fed on a diet poor in lime, but very different to what they observed in cases of spontaneous rickets. The bones in the experimental animals were soft, with some thickening of the epiphyses. The bony trabeculae were merely thinned and did not reveal the presence of any osteoid tissue. In the cases of spontaneous rickets, however, the epiphyses were enormously thickened and much osteoid tissue was present. That it is due to a want of lime is unlikely when one considers that farinaceous foods and cow's milk—the diet on which rachitis is supposed to flourish—contain more than an abundance of that substance. There is no doubt, of course, that during the rachitic processes there is a deficient absorption of lime by the newly-formed bony tissue, and just as truly is there an increased katabolism of the same salts, but it is just on what this perversion of metabolism depends that the crux of the whole question lies.

Hygiene and climate are acknowledged by most authorities to play some part in the etiology of rickets, but only to a slight extent, and in a very general way. To this question, however, I will return later.

The infective theory has been mooted by many, but as yet there is no definite proof in favour of any such idea. Drs. A. Torane and Salvatore Forte[17] believe they have induced the disease in rabbits by inoculating them with watery and alcoholic extracts of the faeces of children suffering from rickets and diarrhoea. Curiously, though rachitic changes are said to follow on injection of either the alcoholic or watery extract, a mixture of the two extracts is quite inactive. Moussu[18] also is of opinion that it is an infectious disease, as he caused a healthy animal to take rickets by confining it in a cage along with a rachitic one; and then, without subsequent disinfection, he confined another healthy animal in the cage, and it also in turn succumbed to the disease. This experimenter, in addition, inoculated various kinds of animals with emulsions of rachitic bone marrow, but without any effect, unless he injected what he calls a very virulent material, and also confined the animals in the above-mentioned, or some other similarly-infected, cage.

Syphilis, too, has been considered answerable, and it is possible that it may act as a predisposing factor; but that it is the prime etiological cause, as Parrot[8] and Marfan[19] consider, is a view which has not received much support in this country, and seems, moreover, to be entirely negatived by the fact that rickets can be easily induced in the lower animals.

The Effects of Deprivation of Exercise.

The great diversity of opinion which prevails regarding the etiology of this disease is shown by the above brief account of the various theories, and is sufficient proof that as yet the true etiological factor has probably not been discovered. Rievel,[20] while summing up the recent work on this subject, suggests that rickets may not have the same origin in all cases, and that many factors may play a part in its causation. It is my intention, however, as the result of some experimental work which I have recently carried out, and by a critical survey of the various influences which affect the child, to show that there is one factor, and that a very potent one, in inducing the disease—namely, want of exercise.

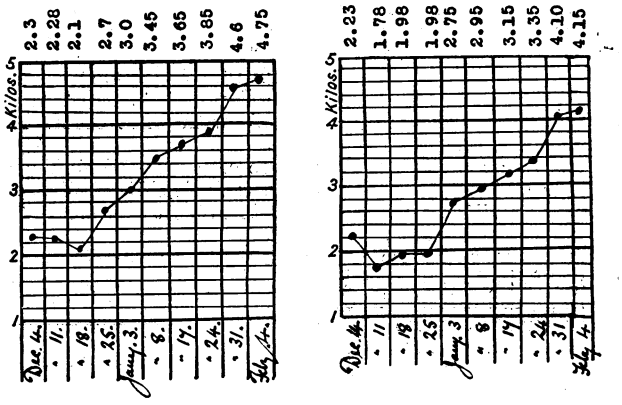
Lack of exercise as a factor in the pathology of the disease has received remarkably little attention, though to my mind it is of the utmost importance, and in future will always require to be taken into consideration when discussing experimental rickets. Though confinement is mentioned by several authors, its mode of action is either misunderstood, or it is supposed to work along with some other baneful influence, and to play a subsidiary part. Dudgeon[21] believes that it acts through lack of fresh air and sunlight, while Clement Lucas[22] mentions it as a contributory factor to incorrect feeding, and Esser[12] to overfeeding. By depriving young dogs of exercise I invariably induced the condition, though they received as much fresh air and sunlight as the control animals. Moreover, their kennels were cleaned regularly. It is not an uncommon experience for experimental animals to develop rickets, consequent, I believe, on the confining of the animals in cages, an almost invariable practice in experimental work. In this way we can explain the case of the rachitic fowl reported by Katz, and also the results of Moussu's experiments. It is in a similar fashion that climate exerts its influence. In warm, genial climates the children will be out much during the day, while in temperate and treacherous climates like our own, especially during the winter, children are exceedingly likely to be cooped up in the house for days, and even weeks, at a time.

It was some two years ago, when commencing an investigation on experimental rickets, that my attention was first attracted to this subject. I attempted to produce rickets in puppies by modified feeding, giving them such generally recognized rachitic diets as bread and water, oatmeal and water, and rice and water, and yet not one developed the disease. They invariably wasted, became marasmic, and died, thus confirming the well-known fact that marasmic infants never become rachitic. But the control animals, at least those which did not become atrophic consequent on diarrhoea, though being fed normally—as, for example, on milk and porridge—all became affected.

In the first series of experiments three control animals developed rickets, one of them, however, less severely than the other two. This animal was fed in a similar fashion to the others, and spent most of its time in their company; but, owing to the fact that it was ultimately intended as a companion, it was exercised by its prospective owner once or twice daily. Accordingly it seemed to me not improbable that in the confinement and want of exercise—the only abnormal factor in the régime of these animals—one had to deal with the etiological factor. I allowed the comparatively healthy animal to get still more exercise for about a week, when all appearances of rickets practically disappeared. On once more confining him with his brothers and sisters he again rapidly became affected. Two of this litter suffered so severely that they were unable to walk, and merely shuffled about the floor of the kennel as if they were affected with paraplegia or diplegia. Similar experiments have been carried out on two other sets of animals, and with exactly the same result. By subjecting the animals to no other abnormal condition than the want of exercise, marked rachitis has invariably ensued.

The following table comprises my observations in chronological order on sixteen animals. Nine of these were treated by confinement and deprived of exercise, but they were fed normally. One (No. 14) died of marasmus, too early, however, to allow of the development of the disease, but the other eight (Nos. 2, 4, 5, 8, 9, 10, 11, and 15) became rachitic to a marked degree. Two (Nos. 1 and 3) were treated by modified dieting and were allowed exercise. Both died of marasmus but without showing any signs of rickets, although they lived beyond the age at which the disease usually appeared in the experiments. The other five (Nos. 6, 7, 12, 13, and 16) were used as controls. They were all allowed exercise, but as regards feeding, housing, and hygiene were treated in a similar fashion to those which developed the disease, and they all remained entirely free of any rachitic manifestation.

I will quote in more detail my last series of experiments. I obtained three collie pups, aged 2 months, from one litter. These three animals were kept in one house, were fed in exactly the same manner on a diet of oatmeal porridge and milk, and were subjected to the same atmospheric conditions as regards amount of sunlight, warmth, and purity of air. Two, however, were confined in a small cage closed on either side but open above, and only covered in front with wire netting, while the other was allowed to run about the room at large and play with a cat. All three at first suffered from diarrhoea, and one of the confined animals in consequence died, but without showing the slightest evidence of rickets. The other two recovered, and steadily put on flesh, and, as seen in the charts A and B, they increased in weight by almost exactly the same amount each week. The puppy allowed to run about developed along normal lines, while the one confined ultimately became rachitic and presented a very typical picture. Fig. 1, from a photograph, shows the contrast between the two animals. The rachitic pup continued to take his food well, and was not troubled with diarrhoea. The first change noticed was a certain degree of languor. When allowed out of the cage he seldom frolicked about, and he was unable, except with the greatest difficulty, to ascend even a very gradual stair. His legs ultimately became bandy, and the beading of the ribs got more marked. His lethargy increased and he lost strength, and, though



A. B.

Charts showing weekly weights of the two animals. A, Healthy dog. B, Rachitic dog.

almost as large and as heavy as his sister, he was frequently overturned by her simply knocking against him. *Post mortem*, the difference between the bones of these two animals was most apparent. In the control the bones were straight and firm, the costo-chondral junctions normal in appearance, and the epiphyseal lines narrow and regular. In the other animal the long bones were curved and exceedingly soft, the epiphyseal lines were broad and irregular (Fig. 2), and there was marked swelling with hyperplasia of the costo-chondral junctions.

In this paper I only intend to deal with the etiology of the condition, and consequently will not devote much space to the pathological changes. In order, however, to show that the condition I had induced experimentally was in reality rickets, it may be advisable to recount the main histological findings in the bones. I contented myself by

Table Showing Results of Experiments.

No.	Sex.	Date when Came Under Observation.	Age when Came Under Observation.		Treatment.	Date when Rickets Developed.†	Cause of Death.	Date of Death.	Time Under Observation, in Weeks.
1	M.	May 11, 1906	At birth	All of one litter	Dietetic	—	Marasmus	Aug. 5, 1906	12
2*	F.	May 11, 1906	,,		Confinement	July 21, 1906	Killed	Aug. 17, 1906	14
3	M.	May 11, 1906	,,		Dietetic	—	Marasmus	Aug. 18, 1906	10
4*	M.	May 11, 1906	,,		Confinement	July 20, 1906	Killed	Aug. 23, 1906	15
5*	M.	May 11, 1906	,,		Modified confinement	July 30, 1906	,,	Aug. 28, 1906	16
6	F.	April 11, 1907	4 weeks	From one litter	Control not confined	—	,,	June 18, 1907	10
7	M.	April 11, 1907	,,		Control not confined	—	,,	June 17, 1907	10
8*	F.	April 11, 1907	5 weeks	From one litter	Confinement	May 14, 1907	,,	June 9, 1907	8½
9*	F.	April 11, 1907	,,		,,	May 14, 1907	,,	June 13, 1907	9
10*	F.	April 11, 1907	,,		,,	May 14, 1907	Bronchopneumonia	June 21, 1907	10
11*	M.	April 11, 1907	4½ weeks		,,	May 12, 1907	,,	June 9, 1907	8½
12	F.	June 3, 1907	8 ,,		Control not confined	—	Killed	July 7, 1907	4½
13	F.	June 3, 1907	9 ,,		Control not confined	—	,,	July 7, 1907	4½
14	M.	Nov. 30, 1907	8 weeks	From one litter	Confinement	—	Marasmus	Dec. 31, 1907	4½
15*	M.	Nov. 30, 1907	,,		,,	Jan. 7, 1908	Killed	Feb. 4, 1908	9
16	F.	Nov. 30, 1907	,,		Control not confined	—	,,	Feb. 4, 1908	9

* Animals which became rachitic.
† These dates of course are only approximate, it being impossible to say exactly from clinical appearances alone when the disease commenced, but at these dates the animals were undoubtedly rachitic.

examining the ribs and long bones. As previously mentioned, the bones were soft and pliable, and in some instances could be flattened between the finger and thumb. This pliancy of the bones was most striking in comparison with the hardness of the bones of normal animals. There was distinct swelling of the epiphyseal ends of the long bones and of the costo-chondral junctions (Fig. 3). Section of the bones revealed enormous thickening of the epiphyseal lines, which measured in some instances 12 mm., and great irregularity and increased vascularity of the same (Fig. 4). Microscopic examination showed much hyperplasia and irregularity of the epiphyseal cartilage—deficient calcification at the growing zone with the development of much osteoid tissue (Fig. 5). The osteoid tissue was, however, more cellular than that occurring in human rickets, but this can perhaps be accounted for by the more rapid development of the disease in the experiments. The little bony tissue present was composed of cells with dwarfed processes irregularly arranged, and contrasted markedly with what occurs in normal bone (Fig. 6).

When we come to correlate these experimental findings with the conditions under which rachitic children live, it will be found that there is much to support this theory of the cause of rickets.

Rickets is a disease of the temperate zone, being very rare, and, in fact, practically unknown, in tropical and sub-tropical countries. The staple diet in these climates is anything but nourishing, the people living mainly on rice, or some other similar cereal, exactly the kind of food which is supposed in this country to generate the disease. The mothers in these countries no doubt live under more natural conditions and suckle their infants; and, as is well known, in Japan, where rickets is extremely rare, and also in Italy, the mothers suckle their children for unduly long periods, and yet with no untoward results. The climate permits these races to spend much of their time in the open air, and the houses are merely used as sleeping apartments. It is this living so much in the open air which probably accounts for their immunity. As previously mentioned, negro and Italian children, though not subject to the disease when reared in their native lands, become almost invariably affected when dwellers in a city, as in New York, where they are said to be the worst-housed people in the States. The fact that rickets occurs chiefly during the winter months lends further support to this theory. It is during the spring that the city dispensaries are crowded with rachitic children, as a result of the winter's confinement. During the winter, in such a climate as ours, a mother, in order to do her child justice, must be on the watch for, and take full advantage of, every dry half-hour during the day—rather an arduous task for even the best disposed of mothers.

It is a disease which is found chiefly among the children of urban populations, being comparatively rare in the country. Further, it is among the children of the poor and the working classes that we find the disease most frequently, and not among those of the rich. One of the main differences between these two classes of children—the rich and the poor—is that the former has usually a nurse to himself, while the latter has to be content with as much or as little spare time as his almost invariably overwrought mother can devote to him. The one is taken out into the fresh air several hours daily, and is entertained at home in an airy nursery, while the other is induced to lie quiet in bed or in some corner of the kitchen, and give as little trouble as possible. The poor man's child undoubtedly gets out little; his mother may have too many children and has no spare time, or she may not perhaps have the inclination to take her child out for an airing. Or it may be that he is the child of some single or widowed woman, who, in order to earn her livelihood, puts him out to a day nursery, where he may be fed and cleaned regularly, but of exercise and entertainment he will receive a minimum. Moreover, the uneducated are notoriously afraid of fresh air in case their children may catch cold by exposure to the weather, so that, with good intention however, the child may not be out for days or weeks at a time, more especially should he be so unfortunate as to suffer from some bronchial catarrh. Again, in cities the poor live in tenements up many flights of stairs, so that there is every inducement for the mother to stay indoors. Well-to-do people find living in tenements a deterrent to going out—how much more, then, must this mode of living affect the irresponsible poor. When the labouring man's child is taken out for a walk, he is almost invariably tightly bandaged to his mother's side with a large shawl, which must impede the movements of his limbs and deprive him of almost all exercise. The great variation in the incidence of this disease among the poor in the country and in the town may perhaps be accounted for by the very different conditions under which they live. The country poor live in cottages, so that the children can be taken out of doors with a minimum of trouble, the very opposite of what prevails in the town.

This confinement of the children, which we find wherever rickets prevails, does of course deprive them of some fresh air and sunlight, and thus reduce their resisting powers, but it is not entirely, or even mainly, on these grounds that it exerts its baneful influence, as my experiments prove. It is due to the want of exercise which invariably goes along with, or is consequent on, the confinement. Children confined to the house are much more lethargic than those taken out of doors. They sleep more, are less vigorous, and are content to lie at rest without evincing any desire for the exercise of their limbs. Every observant parent and physician must have noticed this, and in the case of a healthy child, it is possible from this fact alone to say whether or not he has been out for his usual airing.

As regards feeding, much of the evidence is of an exceedingly doubtful nature. I cannot believe that the poor in the town feed their children so much less intelligently than those in the country as to account for the great preponderance of the disease among their offspring. Their children are often overfed, but is that not equally true of the well-to-do town child? Does the rich parent never overfeed his child, and does the well-to-do mother invariably, or even usually, suckle her infant? As is well known, several observers have been unable to induce rickets experimentally by incorrect feeding. Further, is it not during the summer months, when gastro-enteritis is rife, and there is consequently a diminished absorption of food, that rickets improves, while in the winter time, with no gastro-enteritis, rickets flourishes? It is questionable, too, if a child, deprived of a sufficient supply of fat and proteid, will steadily put on weight, and yet such is not infrequently observed in both experimental and spontaneous rickets. In all my experiments the animals continued to increase in weight, for some time at least after the condition had definitely declared itself.

Rickets is admitted on all hands to be an eminently curable disease, its dangers depending on the susceptibility of the children to pulmonary mischief, and on the deformities which result, especially in the female sex. In treating the condition, the physician often pays as much

DESCRIPTION OF FIGURES IN SPECIAL PLATE.

Fig. 1.—Photograph of the two puppies some ten days before death. Note the extreme degree of bending of the forelegs in the rachitic animal.

Fig. 2.—Photograph of sections of bones of forelegs of the two animals. A, Healthy dog. B, Rachitic dog. Note the bending of humerus, and the great thickening of the epiphyseal lines in the radius and ulna of the rachitic animal.

Fig. 3.—Photograph of inner aspect of the thoracic wall of a healthy and rachitic animal to show the marked rosary in the case of the latter.

Fig. 4.—Longitudinal sections of bones of hind legs in a rachitic, B, and a healthy animal, A, showing the thinning of shafts, and great irregularity and widening of epiphyseal lines in the case of the former.

Fig. 5.—Photomicrograph of a section of rachitic bone stained with thionin blue as advised by Schmorl for the demonstration of bone cells. Note the presence of much osteoid tissue, A, and the dwarfed processes and irregular arrangement of the bone cells, B. C, Marrow.

Fig. 6.—Section of normal bone for comparison. Here there is no osteoid tissue. The bone cells are regularly arranged, and extend right to the margins of the trabeculae. In one trabecula some unabsorbed fetal bone, D, is present. C, Osteoblasts. B, Marrow.

TO ILLUSTRATE DR. LEONARD FINDLAY'S PAPER.

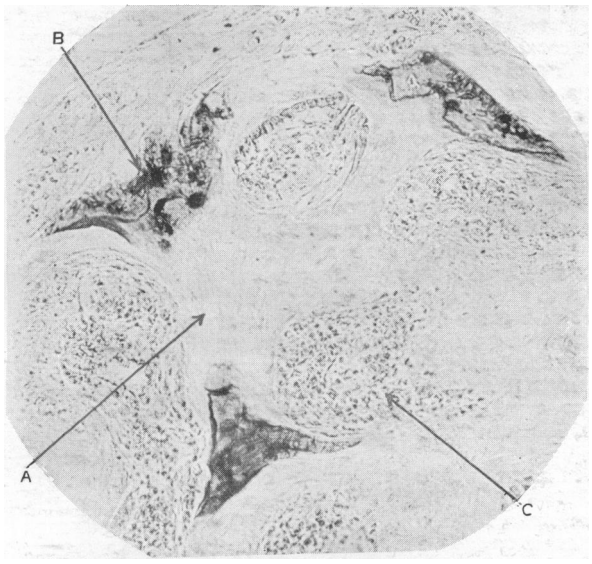


Fig. 5.

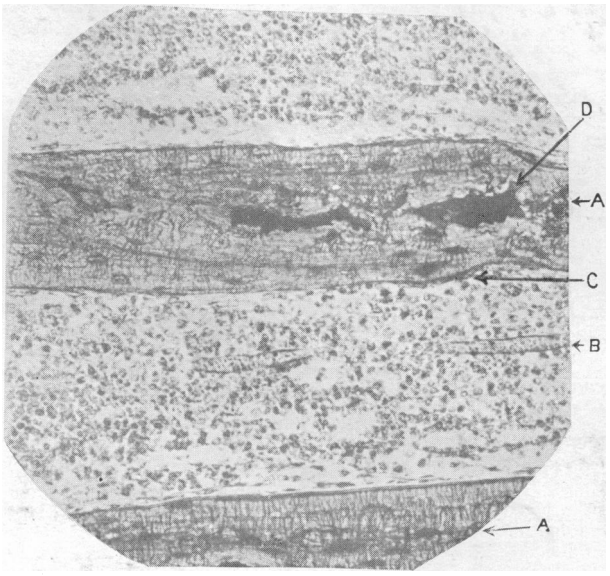


Fig. 6.

Fig. 1.

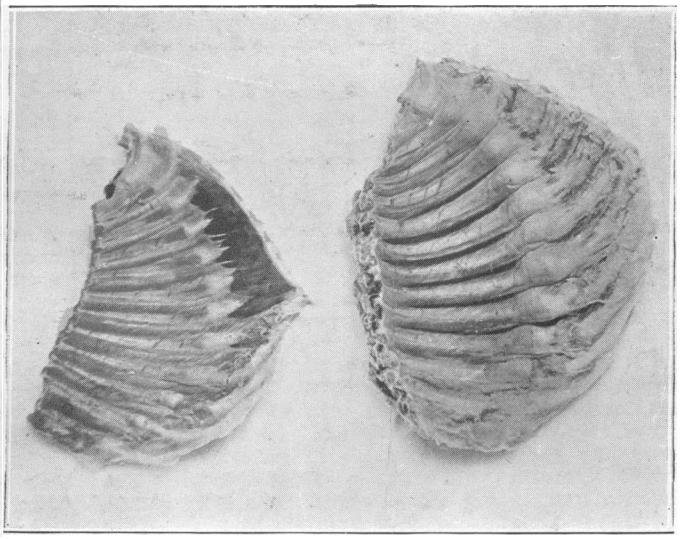


Fig. 3.

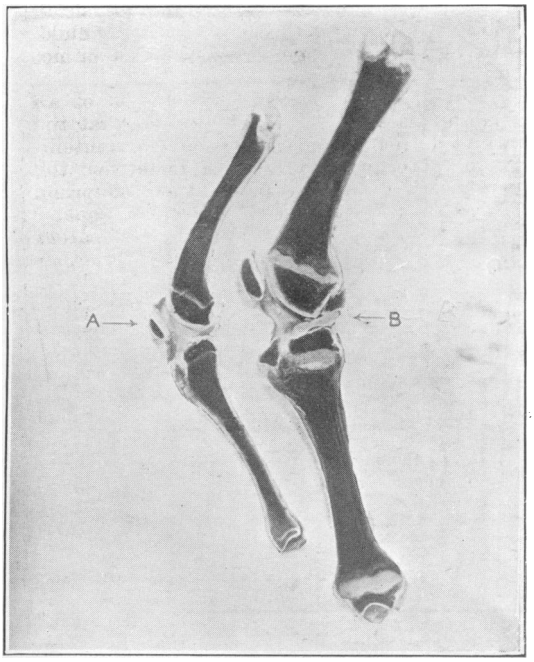


Fig. 4.

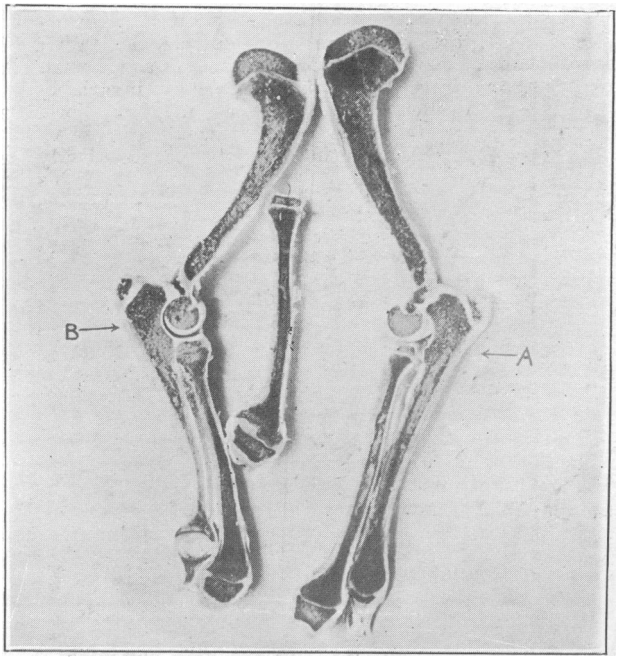


Fig. 2.

attention to the general hygiene as to the question of feeding, and he may at the same time prescribe cod-liver oil and a haematinic, of either of which, however, it is doubtful if much is absorbed. Every one has had the experience of seeing marked improvement take place in two or three weeks. It may not be possible for these poor people completely to revolutionize the hygienic conditions of their homes, but it is certainly possible to take the child out more often into the open air, to give him a daily bath, and to practise a little massage and passive movement of the limbs. Much more easy is it, to my mind, materially to alter the hygiene of the child than the manner of feeding, even to a slight extent. Will a child who has been accustomed for many months to "the run of the house," with all its attendant delicacies, allow much curtailment of its diet, and remain quiet on a simpler and healthier fare? Even should this revolution in feeding take place, will the intestinal mucosa return so speedily to its normal, and allow of perfect absorption? Again, there are cases in which it is impossible to detect any fault in the manner of feeding, though the mother will frankly admit not taking the child out in case it might catch cold. Holt, though he holds that rickets is due to incorrect feeding, admits that he cannot find any error in the dieting of the negro and Italian children in New York.

In the majority of cases a child develops rickets before it has learned to walk, that is, when it is still dependent on its mother and nurse for its exercise. There are many cases, however, in which the child has commenced to walk only to go off its feet later and become markedly rachitic. Perhaps in most of these instances the condition is secondary to some illness, as, for example, measles with bronchitis; and, in order to avoid a recurrence of the complication, the child has been kept indoors, and guarded against the slightest draught of fresh air. But there are other cases in which there is no such history of an intercurrent illness, but owing to the mother being pregnant, or ill from some other cause, the child has not been taken out sufficiently often. In neither of these classes of cases can we call to our aid as a cause the manner of feeding: for months the feeding has been the same, and in many instances such as we cannot take the slightest exception to. In all, however, we get the same story of confinement, and the explanation of the condition lies in the want of exercise.

It is on the ground of the above experiments and facts in the child's history that I am forced to the conclusion that want of exercise is the chief etiological factor in this unfortunately too common malady. It is possible that there may be a toxin responsible for the immediate results, but without lack of exercise this toxin will not produce any injurious effects. We may surmise that the lack of exercise and fresh air may be productive of a certain amount of perversion of metabolism, which allows of the generation of some harmful product, and so by auto-intoxication brings about the disease. But until this specific toxin is isolated and its nature and mode of formation understood, any such idea is mere theorizing and an easy refuge for ignorance. Typhus fever does not arise *de novo*, and yet we know that somehow or other overcrowding allows the disease to proclaim itself, and by legislation against overcrowding this disease has been kept at bay. Overcrowding and typhus fever are no more closely related to one another than lack of exercise and rickets, and by instilling this fact into the minds of mothers, and especially those of the poorer classes, as well as into corporations who have the welfare of the infant race at heart, rickets would undoubtedly become a very rare disease. Far be it from my intention to belittle dietetics, which is one of the most important branches of pediatrics. Faulty feeding is the cause of much of the infantile mortality, but that it plays any important part in the etiology of rickets is very doubtful.

CONCLUSIONS.

1. Not one of the many theories which have been elaborated to explain the cause of rickets has been universally accepted, and they all lack, not only from the clinical but also from the experimental aspect, unequivocal proof.

2. It is some error in feeding which, in this country and America, is commonly believed to bring about the disease, but it is doubtful, however, if feeding plays any important part in the etiology of rickets. Experimentally I, like several other observers, have been unable to cause the condition by improper feeding.

3. By confining young dogs and depriving them of exercise, rickets has been invariably induced, as in the experiments detailed, and that although their diet was beyond suspicion, the air which they breathed pure, and their kennels were kept scrupulously clean, whereas control animals allowed exercise, but otherwise similarly treated, did not become affected.

4. Examination of the conditions under which rachitic children are reared reveals one constant and invariable factor in their lives, namely, confinement. Alike, then, on clinical and experimental grounds I accordingly conclude that confinement, with consequent lack of exercise, is the main factor in causing the disease.

REFERENCES.

[1] Kassowitz, *Die Pathogenese der Rachitis*, Wien, 1885. [2] Tschistowitsch, *Virchow's Archiv*, No. 148, p. 140. [3] Escher, *Jahrb. f. Kinderheilk.*, Bd. 56, p. 613. [4] Hausen, Manuscript f. Dyerläger, xv. [5] Siegert, *Jahrb. f. Kinderheilk.*, Bd. 58, p. 929. [6] Siegert, *Münch. med. Woch.*, 1905, p. 622. [7] Holt, *Diseases of Infancy and Childhood*, 1907, p. 251. [8] Cheadle, *Allbutt's System of Medicine*, 1897, vol. iii, p. 131. [9] Quoted by Cheadle, loc. cit., p. 128. [10] Ashby, *Gibson's Textbook of Medicine*, 1901, p. 491. [11] Heitzman, quoted by Cheadle, loc. cit., p. 130. [12] Esser, *Münch. med. Woch.*, 1907, p. 818. [13] Cheadle, loc. cit., p. 123. [14] Voit, *Zeit. f. Biologie*, xvi, p. 55 (quoted by Rievel, loc. cit.). [15] Katz, *Monats. f. Tierheilk.*, v. i. [16] Reimers and Boye, *Zentralb. f. inn. Med.*, xxvi, H. 39. [17] Iorane and Salvatore Forte, *La Pediatria*, September, 1907. [18] Moussu, *Bull. de la Soc. Centr.*, lvii. [19] Marfan, *La Semaine Médicale*, 1907, No. 40. [20] Rievel, Lubarsch-Ostertag, 1907, Bd. ii, p. 609. [21] Dudgeon, *Rep. of the Soc. for Study of Dis. in Child.*, vol. vii, p. 56. [22] Clement Lucas, *Brit. Journ. of Child. Dis.*, February, 1908.

CLINICAL NOTES ON SOME CAUSES OF PERITONITIS OCCURRING DURING THE COURSE OF PREGNANCY.

BY ARCHIBALD CUFF, M.B., B.C., F.R.C.S.,

SURGEON TO THE ROYAL INFIRMARY, SHEFFIELD.

CONDITIONS likely to produce peritoneal inflammation are fortunately not of common occurrence in pregnant women, for when they do arise their course is often more severe, their prognosis graver, and they are likely to seriously affect the pregnancy; so, too, they are more easily overlooked, and their diagnosis is in all cases arrived at with greater difficulty.

The more usual causes of peritonitis in women are appendicitis, salpingitis, rupture, complete or partial, of ulcers of the stomach, inflammatory changes in cysts of the ovary with or without torsion of their pedicles, and degenerative changes in uterine fibroids. Peritonitis due to salpingitis, though of extreme frequency in the non-pregnant state, is but rarely seen during pregnancy. The reason for this is probably that salpingitis diminishes the possibility of pregnancy.

Gastric ulcers seem to be of infrequent occurrence during pregnancy, though some years ago I saw a fatal case of general peritonitis due to the rupture of an ulcer in a pregnant woman.

The special interest in all causes of peritonism arising in pregnancy is dependent mainly on the variations in the clinical course which the process is likely to run, especially with reference to (1) the symptoms, complications, and prognosis, (2) the effect of the disease on the pregnancy, and (3) the special difficulties in arriving at a correct diagnosis of the particular lesion present. With regard to the effect on the pregnancy, we have to consider on one hand the effect of the morbid process itself, and on the other the effect of the necessary surgical treatment.

There is, I think, no doubt that shock produced by physical or mental processes may lead to abortion, even if there be no interference with the uterus or abdomen; much more so is this likely to be the case if an abdominal operation is necessary. Especially is the effect to be feared in women of very nervous temperament. While, therefore, it is unwise to perform an operation of any importance during pregnancy, except in the case of urgent necessity, it is possible that with greater experience and with modern methods and technique the fear expressed by the older generation of surgeons of operating during pregnancy will be shown to be in great measure unfounded.

Certainly to-day major operations of urgency are done again and again, without ill effect, and I have myself on divers occasions operated for such diseases as gall stones, hydatid of the liver, renal calculus, and for ovarian cyst,